IN THE CLAIMS:

Please cancel claims 17, 52, 60 and 88 to 126 without prejudice.

1. (Original) A method of increasing glycogen to toxic levels in a cell, comprising expressing in the cell a gene product that increases the amount of glycogen to toxic levels in the cell.

2. (Original) The method of claim 1, wherein the gene product comprises a protein that increases synthesis or intracellular accumulation of glycogen.

3. (Original) The method of claim 1, wherein the gene product comprises a protein that decreases glycogen metabolism, catabolism, utilization, degradation or removal.

4. (Original) The method of claim 1, wherein the glycogen is in an amount that causes a morphological change associated with glycogen toxicity.

5. (Original) The method of claim 4, wherein the morphological change associated with glycogen toxicity comprises cell swelling, increased numbers of lysosomes, increased size of lysosomes, or a structural change in lysosomes.

6. (Original) The method of claim 1, wherein the glycogen is in an amount that causes lysis or apoptosis of the cell.

7. (Original) The method of claim 1, wherein the glycogen is in an amount that inhibits or reduces proliferation, growth or survival of the cell.

8. (Original) The method of claim 1, wherein the gene product comprises a glycogenic enzyme.

9. (Original) The method of claim 1, wherein the glycogenic enzyme comprises glycogenin, glycogenin-2, glycogen synthase, glycogenin interacting protein (GNIP), protein phosphatase 1 (PP-1), glucose transporter (GLUT), a glycogen targeting subunit of PP-1 isoform or family member, a hexokinase isoform or family member, or glutamine-fructose-6-phosphate transaminase.

10. (Original) The method of claim 9, wherein the glycogen targeting subunit of PP-1 family member comprises G_L (PPP1R3B, PPP1R4), PTG (PPP1R3C, PPP1R5), PPP1R3D (PPP1R6) or G_m/R_{G1} (PPP1R3A, PPP1R3).

11. (Original) The method of claim 1, wherein the gene product comprises an antisense polynucleotide, a small interfering RNA molecule, or a ribozyme that reduces expression of a glycogenolytic enzyme.

12. (Original) The method of claim 11, wherein the glycogenolytic enzyme comprises glycogen phosphorylase, debranching enzyme, phosphorylase kinase, glucose-6-phosphatase, PPP1R1A (protein phosphatase 1, regulatory Inhibitor subunit 1A), PPP1R2 (protein phosphatase 1, regulatory subunit 2), phosphofructokinase, a glycogen synthase kinase-3 isoform, GCKR glucokinase regulatory protein or α-glucosidase.

13. (Original) The method of claim 1, wherein the cell comprises a hyperproliferative cell.

14. (Original) The method of claim 13, wherein the hyperproliferative cell comprises a metastatic or non-metastatic cancer cell.

15. (Original) The method of claim 14, wherein the cancer cell is present in brain, head or neck, breast, esophagus, mouth, stomach, lung, gastrointestinal tract, liver, pancreas, kidney, adrenal gland, bladder, colon, rectum, prostate, uterus, cervix, ovary, testes, skin, muscle or hematopoetic system.

16. (Original) The method of claim 14, wherein the hyperproliferative cell is present in a subject.

17. (Canceled)

18. (Original) The method of claim 16, wherein the subject is a human.

19. (Original) The method of claim 1, wherein the gene product comprises a protein, an antisense polynucleotide, a small interfering RNA or a ribozyme.

20. (Original) The method of claim 1, wherein the gene product is encoded by a polynucleotide.

21. (Original) The method of claim 20, wherein the polynucleotide comprises a vector.

22. (Original) The method of claim 20, wherein the vector comprises a viral or mammalian expression vector.

23. (Original) The method of claim 20, wherein the polynucleotide further comprises a vesicle.

24. (Original) The method of claim 1, wherein expression of the gene product is conferred by a promoter active in a hyperproliferative cell.

25. (Original) The method of claim 24, wherein the promoter comprises hexokinase II, COX-2, alpha-fetoprotein, carcinoembryonic antigen, DE3/MUC1, prostate specific antigen, C-erB2/neu, telomerase reverse transcriptase or hypoxia-responsive promoter.

26. (Original) The method of claim 1, further comprising expressing in the cell a second protein that inhibits cell proliferation.

27. (Original) The method of claim 26, wherein the second protein comprises a cell cycle inhibitor.

28. (Original) The method of claim 26, wherein the second protein comprises a cyclin inhibitor.

29. (Original) A method of increasing glycogen to toxic levels in a hyperproliferative cell, comprising contacting the cell with an agent that increases the amount of glycogen to toxic levels in the hyperproliferative cell, wherein the hyperproliferative cell is not a liver, muscle or brain cell.

30. (Original) The method of claim 29, wherein the glycogen is in an amount that causes a morphological change associated with glycogen toxicity.

31. (Original) The method of claim 29, wherein the glycogen is in an amount that causes lysis or apoptosis of the cell.

32. (Original) The method of claim 29, wherein the glycogen is in an amount that inhibits or reduces proliferation, growth or survival of the cell.

33. (Original) The method of claim 29, wherein the agent increases expression or activity of a glycogenic enzyme.

34. (Original) The method of claim 33, wherein the glycogenic enzyme is selected from glycogenin, glycogenin-2, glycogen synthase, glycogenin interacting protein (GNIP), protein phosphatase 1 (PP-1), glucose transporter (GLUT), a glycogen targeting subunit of PP-1 isoform or family member, a hexokinase isoform or family member, or glutamine-fructose-6-phosphate transaminase.

35. (Original) The method of claim 29, wherein the agent decreases expression or activity of a glycogenolytic enzyme.

36. (Original) The method of claim 29, wherein the agent comprises an antisense, ribozyme, siRNA or triplex forming nucleic acid that specifically binds to a glycogenolytic enzyme.

37. (Original) The method of claim 35, wherein the glycogenolytic enzyme is selected from glycogen phosphorylase, debranching enzyme, phosphorylase kinase, glucose-6-phosphatase, PPP1R1A (protein phosphatase 1, regulatory Inhibitor subunit 1A), PPP1R2 (protein phosphatase 1, regulatory subunit 2), phosphofructokinase, a glycogen synthase kinase-3 isoform, GCKR glucokinase regulatory protein or α-glucosidase.

38. (Original) A method of increasing glycogen to toxic levels in a hyperproliferative cell, comprising contacting the cell with an agent that increases the amount of glycogen to toxic levels in the hyperproliferative cell, provided that the agent does not substantially inhibit activity or expression of a glycogen phosphorylase isotype.

39. (Original) The method of claim 38, wherein the glycogen phosphorylase isotype comprises a liver, muscle or brain glycogen phosphorylase.

40. (Original) The method of claim 38, wherein the glycogen is in an amount that causes a morphological change associated with glycogen toxicity.

41. (Original) The method of claim 38, wherein the glycogen is in an amount that causes lysis or apoptosis of the cell.

42. (Original) The method of claim 38, wherein the glycogen is in an amount that inhibits or reduces proliferation, growth or survival of the cell.

43. (Original) The method of claim 38, wherein the agent increases expression or activity of a glycogenic enzyme.

44. (Original) The method of claim 43, wherein the glycogenic enzyme is selected from glycogenin, glycogenin-2, glycogen synthase, glycogenin interacting protein (GNIP), protein phosphatase 1 (PP-1), glucose transporter (GLUT), a glycogen targeting subunit of PP-1 isoform or family member, a hexokinase isoform or family member, or glutamine-fructose-6-phosphate transaminase.

45. (Original) The method of claim 38, wherein the agent decreases expression or activity of a glycogenolytic enzyme.

46. (Original) The method of claim 38, wherein the agent comprises an antisense, ribozyme, sIRNA or triplex forming nucleic acid that specifically binds to a glycogenolytic enzyme.

47. (Original) The method of claim 45, wherein the glycogenolytic enzyme is selected from debranching enzyme, phosphorylase kinase, glucose-6-phosphatase, PPP1R1A (protein phosphatase 1, regulatory Inhibitor subunit 1A), PPP1R2 (protein phosphatase 1, regulatory subunit 2), phosphofructokinase, a glycogen synthase kinase-3 isoform, GCKR glucokinase regulatory protein or α-glucosidase.

48. (Original) The method of claim 38, wherein the agent comprises a substrate analogue.

49. (Original) The method of claim 38, wherein the hyperproliferative cell comprises a metastatic or non-metastatic cancer cell.

50. (Original) The method of claim 49, wherein the cancer cell is present in brain, head or neck, breast, esophagus, mouth, stomach, lung, gastrointestinal tract, liver, pancreas, kidney, adrenal gland, bladder, colon, rectum, prostate, uterus, cervix, ovary, testes, skin or muscle, or hematopoetic system.

51. (Original) The method of claim 38, wherein the hyperproliferative cell is present in a subject.

52. (Canceled)

53. (Original) The method of claim 51, wherein the subject is a human.

54. (Original) A method of treating a cell proliferative disorder in a subject, wherein the cell proliferative disorder is not a liver, muscle or brain cell disorder, comprising expressing in one or more cells comprising the disorder a gene product that increases the amount of intracellular glycogen, or comprising contacting one or more cells comprising the disorder with an agent that increases the amount of intracellular glycogen, sufficient to treat the cell proliferative disorder.

55. (Original) The method of claim 54, wherein the cell proliferative disorder comprises a metastatic or non-metastatic cancer.

56. (Original) The method of claim 55, wherein the cancer cell is present in head or neck, breast, esophagus, mouth, stomach, lung, gastrointestinal tract, pancreas, kidney, adrenal gland, bladder, colon, rectum, prostate, uterus, cervix, ovary, testes, skin, or hematopoetic system.

57. (Original) A method of treating a cell proliferative disorder of a subject, comprising expressing in one or more cells comprising the disorder a gene product that increases the amount of intracellular glycogen, or comprising contacting one or more cells comprising the disorder with an agent in an amount that increases the amount of intracellular glycogen, provided that the agent does not substantially inhibit activity or expression of a glycogen phosphorylase isotype, sufficient to treat the cell proliferative disorder.

58. (Original) The method of claim 57, wherein the cell proliferative disorder comprises a metastatic or non-metastatic cancer.

59. (Original) The method of claim 58, wherein the cancer cell is present in brain, head or neck, breast, esophagus, mouth, stomach, lung, gastrointestinal tract, liver, pancreas, kidney, adrenal gland, bladder, colon, rectum, prostate, uterus, cervix, ovary, testes, skin or muscle, or hematopoetic system.

60. (Canceled)

61. (Original) The method of claims 54 and 57, wherein the subject is human.

62. (Original) A method of treating a subject having a tumor, wherein the tumor is not a liver, muscle or brain tumor, comprising expressing in one or more of the tumor cells a gene product that increases the amount of intracellular glycogen, or comprising contacting one or more of the tumor cells with an agent that increases the amount of intracellular glycogen, effective to treat the subject.

63. (Original) A method of treating a subject having a tumor, comprising expressing in one or more of the tumor cells a gene product that increases the amount of intracellular glycogen, or comprising contacting one or more of the tumor cells with an agent in an amount that increases the amount of intracellular glycogen, provided that the agent does not substantially inhibit activity or expression of a glycogen phosphorylase isotype, effective to treat the subject.

64. (Original) A method of treating a subject that is undergoing or has undergone tumor therapy, wherein the tumor therapy was not for a liver, muscle or brain tumor, comprising administering to the subject an agent in an amount that increases the amount of intracellular glycogen in a cell sufficient to treat the subject.

65. (Original) A method of treating a subject that is undergoing or has undergone tumor therapy, comprising administering to the subject an agent in an amount that increases the

amount of intracellular glycogen, provided that the agent does not substantially inhibit activity or expression of a glycogen phosphorylase isotype, sufficient to treat the subject.

66. (Original) A method of increasing effectiveness of an anti-tumor therapy, comprising administering to a subject that is undergoing or has undergone anti-tumor or immune-enhancing therapy, wherein the tumor therapy was not for a liver, muscle or brain tumor, an agent in an amount that increases the amount of intracellular glycogen, and an anti-tumor or immune-enhancing therapy.

67. (Original) A method of increasing effectiveness of an anti-tumor therapy, comprising administering to a subject that is undergoing or has undergone anti-tumor or immune-enhancing therapy, an agent in an amount that increases the amount of intracellular glycogen, provided that the agent does not substantially inhibit activity or expression of a glycogen phosphorylase isotype, and an anti-tumor or immune-enhancing therapy.

68. (Original) The method of any of claims 66 or 67, wherein the agent is administered prior to, substantially contemporaneously with or following administration of the anti-tumor or immune-enhancing therapy.

69. (Original) The method of any of claims 62 to 67, wherein the tumor comprises a metastatic or non-metastatic tumor.

70. (Original) The method of any of claims 62 to 67, wherein the tumor comprises a stage I, II, III, IV or V tumor.

71. (Original) The method of any of claims 62 to 67, wherein the tumor is in remission.

72. (Original) The method of any of claims 62 to 67, wherein the tumor is solid or liquid.

73. (Original) The method of any of claims 62, 64 and 66, wherein the tumor is located at least in part in head or neck, breast, esophagus, mouth, stomach, lung, gastrointestinal tract, pancreas, kidney, adrenal gland, bladder, colon, rectum, prostate, uterus, cervix, ovary, testes, or skin.

74. (Original) The method of any of claims 63, 65 and 67, wherein the tumor is located at least in part in brain, head or neck, breast, esophagus, mouth, stomach, lung, gastrointestinal tract, liver, pancreas, kidney, adrenal gland, bladder, colon, rectum, prostate, uterus, cervix, ovary, testes, skin or muscle.

75. (Original) The method of any of claims 62 to 67, wherein the tumor is haematopoetic.

76. (Original) The method of any of claims 62 to 67, wherein the tumor comprises a sarcoma, carcinoma, melanoma, myeloma, blastoma, glioma, lymphoma or leukemia.

77. (Original) The method of any of claims 62 to 67, wherein the treatment reduces tumor volume, inhibits an increase in tumor volume, inhibits progression of the tumor, stimulates tumor cell lysis or apoptosis, or inhibits tumor metastasis.

78. (Original) The method of any of claims 62 to 67, wherein the treatment reduces one or more adverse symptoms associated with the tumor.

79. (Original) The method of any of claims 62 to 67, wherein the treatment prolongs lifespan of the subject.

80. (Original) The method of any of claims 62 to 67, wherein the subject is a candidate for, is undergoing, or has undergone anti-tumor or immune-enhancing therapy.

81. (Original) The method of any of claims 62 to 67, further comprising administering an anti-tumor or immune enhancing treatment or agent.

82. (Original) The method of claim 81, wherein the anti-tumor treatment comprises chemotherapy, immunotherapy, surgical resection, radiotherapy or hyperthermia.

83. (Original) The method of claim 81, wherein the anti-tumor agent comprises an alkylating agent, anti-metabolite, plant extract, plant alkaloid, nitrosourea, hormone, nucleoside or nucleotide analogue.

84. (Original) The method of claim 81, wherein the anti-tumor agent is selected from: cyclophosphamide, azathioprine, cyclosporin A, prednisolone, melphalan, chlorambucil,

mechlorethamine, busulphan, methotrexate, 6-mercaptopurine, thioguanine, 5-fluorouracil, cytosine arabinoside, AZT, 5-azacytidine (5-AZC) and 5-azacytidine related compounds, bleomycin, actinomycin D, mithramycin, mitomycin C, carmustine, lomustine, semustine, streptozotocin, hydroxyurea, cisplatin, mitotane, procarbazine, dacarbazine, taxol, vinblastine, vincristine, doxorubicin and dibromomannitol.

85. (Original) The method of claim 81, wherein the immune enhancing treatment comprises administration of a lymphocyte, plasma cell, macrophage, dendritic cell, NK cell or B-cell.

86. (Original) The method of claim 81, wherein the immune enhancing agent comprises an antibody, a cell growth factor, a cell survival factor, a cell differentiative factor, a cytokine or a chemokine.

87. (Original) The method of claim 81, wherein the immune enhancing agent is selected from: IL-2, IL-1α, IL-1β, IL-3, IL-6, IL-7, granulocyte-macrophage-colony stimulating factor (GMCSF), IFN-γ, IL-12, TNF-α, TNFβ, MIP-1α, MIP-1β, RANTES, SDF-1, MCP-1, MCP-2, MCP-3, MCP-4, eotaxin, eotaxin-2, I-309/TCA3, ATAC, HCC-1, HCC-2, HCC-3, LARC/MIP-3α, PARC, TARC, CKβ, CKβ6, CKβ7, CKβ8, CKβ9, CKβ11, CKβ12, C10, IL-8, GROα, GROβ, ENA-78, GCP-2, PBP/CTAPIIIβ-TG/NAP-2, Mig, PBSF/SDF-1, and lymphotactin.

88.-126. (Canceled)